

# Deafness

Deafness means inability to hear and is of four types, namely:-

1. Conductive.
2. SNHL
3. Mixed (conductive and perceptive).
4. Non-organic (hysterical and feigned).

Causes of conductive deafness

1. Causes in the external ear.
2. Causes in the middle ear.

A) Congenital

1. Crouzon's syndrome (Craniofacial dysostosis). Conductive deafness is due to external canal atresia & middle ear abnormalities.
2. Wildervanck syndrome, in which a conductive deafness is due to fusion or other malformation of the ossicular chain is associated with pre-auricular sinuses, pre-auricular appendages and malformation of the auricle.
3. Treacher Collins syndrome. An hereditary malformation of the lower face in which the malar bones, maxillae and mandible are hypoplastic, together with varying degrees of developmental failure of the external and middle ears. It may be unilateral or bilateral.

B) Injuries

1. Foreign bodies.
2. Traumatic rupture of the tympanic membrane.
3. Fracture of the temporal bone (longitudinal type).
4. Traumatic disconnection of ossicular chain.
5. Barotraumatic otitis media.

C) Otitis media. (non-specific and specific, acute and chronic, secretory or suppurative)

D) Neoplasms of the middle ear cleft.

1. Benign: glomus tumour (chemodectoma)
2. Malignant: squamous cell carcinoma.

E) Miscellaneous

1. Otosclerosis.
2. Paget's disease of the temporal bone.
3. Osteogenesis imperfecta.
4. Histocytosis-X.

## **Sensori-neural Hearing Loss**

This type of deafness can occur due to lesion in cochlea, auditory nerve or in auditory pathways.

### **Causes**

#### **1. Congenital**

- **Alport syndrome.** Associated with hereditary familial, congenital haemorrhagic nephritis.
- **Pendred syndrome.** Associated with sporadic goiter.

#### **2. Traumatic**

- **Blast injury.**
- **Temporal bone fracture.**
- **Noise induced hearing loss.**

#### **3. Infections**

- **Viral (small pox, mumps, measles, herpes zoster)**
- **Bacterial (meningococcal meningitis & encephalitis, TB, syphilis, typhoid fever).**

#### **4. Ototoxicity (quinine, aminoglycosides, diuretics, aspirin).**

#### **5. Metabolic (hypothyroidism, avitaminosis, diabetes mellitus).**

#### **6. Presbycusis (senile deafness)**

#### **7. Meniere's disease**

#### **8. Vestibular schwannoma**

#### **9. Vascular insufficiency or ischemia of cochlea**

**Internal auditory artery is an end artery & its spasm, thrombosis & haemorrhage cause damage to hearing.**

### **Clinical Features**

#### **Symptoms**

- Hearing loss may be unilateral or bilateral depending on the cause.
- Distortion of hearing.
- Unusual difficulty created by background noise.
- Impaired discrimination.
- Difficulty in localizing the sound.
- Tinnitus is often troublesome.

#### **On examination**

- The tympanic membrane will generally be normal unless there has been some middle ear problem.
- Tuning fork tests (Rinne test positive, Weber test lateralized to the better or normal side, ABC will be shortened).

#### **Treatment**

1. Treat the cause if possible.
2. Hearing aid.
3. Cochlear implant.

# Tinnitus

**Definition:** A sensation of noises in the ear or head. The term does not include hallucinations of voices, which are of psychogenic origin.

## Types

1. Subjective: which is the usual type.
2. Objective: clicks or buzzing may be heard by the observer, vascular bruit must be excluded.

## Causes

Almost every ear disease and cause of deafness can be associated with tinnitus, and the great majority of tinnitus sufferers have some measurable hearing loss. There is much evidence relating the prevalence and severity of tinnitus to the amount of hearing loss.

### **1. Local Causes.**

- **Presbycusis.**
- **Noise-induced hearing loss.**
- **Meniere's disease.**
- **Otosclerosis.**
- **Vestibular schwannoma.**

### **2. General Causes**

- **Cardiovascular disease (hypertension, cardiac failure).**
- **Blood disease (anaemia, raised viscosity).**
- **Neurological (multiple sclerosis, neuropathy).**
- **Drugs (aspirin, quinine, ototoxic drugs).**
- **Alcohol abuse.**
- **Fever of any cause.**

## Clinical Assessment

From the patient's history confirm that the patient is actually suffering with tinnitus and determine the character of the sound (intermittent or constant, pulsatile or non-pulsatile). It is also important to establish how much trouble the patient is having because this will dictate whether treatment is necessary.

A full ENT examination should be performed to ensure the patient is not hearing transmitted sounds or does not have one of the remedial causes of tinnitus. The ears should be examined under the microscope. Small movements of the tympanic membrane are sometimes visible (secondary to tensor tympani myoclonic contractions or abnormal patency of the Eustachian tube) & glomus jugulare tumours may be visualized. Auscultate the neck, head & ears with stethoscope for bruits or myoclonus. The blood pressure and pulse should also be measured.

## Investigations

1. In the absence of a clear diagnosis perform a full haematological screen to exclude anaemia, thyroid dysfunction, syphilis, dyslipidaemia & hypoglycaemia.
2. PTA: to determine the type and severity of deafness.
3. In patients with markedly asymmetrical tinnitus, it should be remembered that 10% of vestibular schwannoma, present in this way. Brainstem electric response audiometry should be requested unless the hearing loss is more than 70dB, when MRI scanning is required.

4. Pulsatile tinnitus may require angiography if a specific vascular lesion is suspected.

### **Treatment;**

1. Any underlying cause should be treated.
2. Hearing aids. These are only useful to those patients who have a 35 dB loss over the speech frequency range. If an appropriate aid with maximal gain at around the frequency of the tinnitus is fitted, the increased awareness of the background sound will make the tinnitus less apparent.
3. Maskers. These produce a controllable and more tonal masking sound which obliterates the sensation of the patient's own tinnitus.
4. Combined hearing aid and masker unit.
5. A pillow radio or pillow speaker may help the patient get to sleep.
6. Many other treatments have been tried (i.e. lignocaine, ultrasonic stimulation, hypnotherapy, dietary supplements, electrical suppression), but success has been limited and adverse effects common.

### **Vertigo**

Definition: vertigo is subjective sensation of imbalance. It is the cardinal symptom of disease of the vestibular system including its central connections.

#### **Causes**

- **Physiological.**
  1. Sea sickness.
  2. Airsickness.
  3. Travel sickness.
  4. Rotation.
  5. Standing on height.
- **Pathological**
  1. Vestibular
- **Peripheral**
  1. Benign positional vertigo commonly occurs after a head injury & is rotatory vertigo with a particular head movement, by the Hallpike manoeuvre.
  2. Meniere's disease comprises paroxysmal fluctuating hearing loss, vertigo, & tinnitus, each attack lasting minutes or hours.
  3. Acute vestibular neuritis consists of marked vertigo for many hours or days often preceded by an URTI.
  4. Vestibular schwannoma usually presents with unilateral sensory hearing loss, but this is often accompanied by tinnitus & occasionally there is a non-specific dizziness.
  5. Middle ear disease such as cholesteatoma.
  6. Drugs (aminoglycosides, diuretics, co-trimoxazole, metronidazole).
  7. Cervical vertigo.
- **Central**
  1. Cerebrovascular disease
  2. Migraine.
  3. Multiple sclerosis.
  4. Brain tumours.
  5. Cerebrovascular insufficiency.

## **2. Non-vestibular**

- **Cardiovascular diseases.**
- **Metabolic diseases.**
- **Haematological diseases.**
- **Musculoskeletal diseases.**

### **Clinical Features**

It is often difficult for patients to describe their sensations, and in taking a full and accurate history the feeling of vertigo must be differentiated from other types of dizziness such as fainting or light-headedness. A full description of the sensation should be obtained with reference to precipitating factors (e.g. neck movements), associated symptoms (e.g. deafness, tinnitus) and frequency and duration of the attacks. A previous history of trauma should be noted. Previous medical history, medication, and alcohol ingestion should also be considered in the context of possible causes or aggravation of the symptoms.

An otological and neurological examination is mandatory in all cases of vertigo. In particular middle-ear disease is looked for by nystagmus after finger following or after Hallpike test. Gait assessment including Romberg's and unterburger testing is important. A general medical examination may be required if the symptoms dictate.

### **Investigations**

Vestibular testing consists of PTA, evoked response audiometry, electronystagmography with caloric stimulation. MRI with gadolinium enhancement is the radiological investigation of choice.

### **Treatment**

1. Vestibular rehabilitation. This is now considered to be the mainstay of treatment in many vestibular disorders.
2. Medical treatment. This consists of lifestyle changes (e.g. less alcohol) and drugs. The latter are usually vestibular sedatives such as prochlorperazine or cinnarizine, histamine analogues such as betahistine, or antidepressants.
3. Surgery. This is usually used for episodic peripheral vertigo diagnosed as Meniere's disease and consists of endolymphatic sac shunting. Vestibular neurectomy or labyrinthectomy. Occasionally it is used for benign positional vertigo when posterior semicircular canal obliteration or singular neurectomy.